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SUBACUTE SPINAL PARALYSIS (PROGRESSIVE)—CASE WITH AUTOPSY.¹

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THE history of subacute spinal paralysis will have to be rewritten, in the light of recent discoveries regarding multiple neuritis. At present, the cases heretofore regarded as of this kind may be divided into three classes.

- I. Those of subacute multiple neuritis.
- II. Those having a course like that of a subacute corneal myelitis, with a tendency to recover—subacute regressive spinal paralysis.
- III. Those having a similar course at first to the preceding, but tending to progress, and develop finally a course like that of progressive muscular atrophy—*subacute progressive spinal paralysis*.

This classification, which is given by Gowers, will doubtless have to be changed in time. The following case illustrates the fact that a case, starting out clinically

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like a form of subacute cornual myelitis, progressed steadily until death.

For the case and specimen, I am indebted to my friend, Dr. Hermann M. Biggs.

Anton S., tailor, æt. 53; nat. Austria.

Admitted to Ward 18, Bellevue Hospital, Aug. 24th, 1886, in Dr. Biggs' service.

History of patient previous to present sickness negative. Patient had never been sick before; family history good. No history of syphilis.

On admission: Patient large, well nourished, and apparently had lost no flesh. Physical examination of chest and abdomen negative.

Pat. had lost almost entirely power over his lower limbs. He could raise them from the bed when lying on his back, but could not support himself on his legs and, if left standing unassisted, would fall immediately to the floor. There was also marked loss of power in the upper extremities, but not so great as in the lower. There did not seem to be any more inco-ordination in his movements than would result from the loss of power over the muscles. There was complete loss of tendon reflexes. There was a moderate degree of general anæsthesia in the lower extremities, with one or two spots of hyperæsthesia on the inner surface of either leg. The action of bladder and rectum was normal, and had been throughout his sickness.

Patient said that he first noticed a weakness in his legs four or five months before his admission; said that this gradually grew worse until about one month before he entered the hospital, when he was obliged to give up work and remain most of the time in bed. Said he had had no pain at any time, and complained of nothing excepting the loss of power in his limbs. There was no affection of any of the special nerves or of cranial nerves. The batteries at the hospital were all out of order, and no electrical reactions could be tested.

Examination of urine negative.

Patient was placed on large doses of iodide of potas-

sium. He, however, gradually lost more and more power over his extremities, but otherwise remained in same general condition until about two days before his death, when he developed a slight fever, became delirious, failed rapidly, and died in partial coma on Sept. 21st, not quite one month after his admission.

At the *autopsy*, nothing of importance was found in any of the thoracic or abdominal viscera. Brain showed nothing. There was a deep injection of the small vessels of the pia mater. There were no lesions in the cord or its membranes visible to the naked eye, except a small cavity about one-twelfth by one-sixteenth inch in diameter in the right half of the intermediate gray and right cornu. The cavity extended from about the sixth cervical to the third or fourth dorsal nerve.

Microscopical examination.—The cord was hardened in Müller's fluid in an incubator. After ten days it was placed in alcohol. Sections were made in the middle and lower cervical region, upper and middle dorsal, and the middle lumbar and sacral segments.

The lower cervical and upper dorsal sections showed the upper limit, the middle part, and the lower limit of the cavity described above.

Middle lumbar region (3d to 4th).--The white matter shows a considerable number of small colloid bodies, especially in the peripheral part of the lateral and posterior columns. These bodies indicate a shrinking or total loss of the nerve fibre,

Large spaces in the posterior columns indicate excessively dilated blood-vessels which have been torn away. In the lateral columns are numerous vessels with decidedly thickened walls. One may count with a two-third-inch objective twelve distinctly-marked distended vessels in the left lateral column, ten in the right. There are eight large spaces made by vessels torn away in the posterior column.

In sections of healthy cords, I have observed about three-fourths this amount of vascularity.

Gray Matter.—The antero-lateral group of cells is

shrunken, and has nearly disappeared on one side. The central group is also affected.

The other anterior cornual groups are not markedly changed. One may observe several large multipolar cells in the central part of the posterior gray cornua.

The gray matter is richly studded with distended capillaries and small vessels. The vascularization in the posterior horns is striking. The vessel walls are not notably thickened. The central canal shows nothing peculiar.

The *middle dorsal region* shows the same changes as above. The colloid bodies referred to, however, are still more numerous. The vascularization of the gray matter is very marked, and there are very few healthy cells in

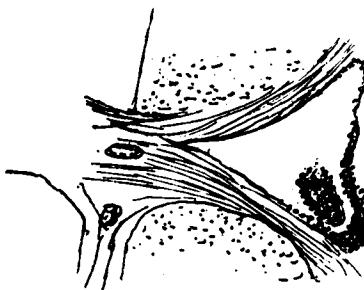


FIG. 1.—A cavity in central gray of lower cervical region, showing blood-vessel and cell infiltration. $\times 400$.

the anterior horns. The cells of Clark's column are shrunken or gone, and there is much vascularity in this region and in the intermediate gray anteriorly to it. The fine nerve mesh-work brought out by Weigert's stain in lower sections is very indistinct here.

Sections in the lower dorsal region show Clark's columns to be normal.

Sections in the sacral segments show nothing abnormal.

Sections in the middle cervical region show a capillary injection of the intermediate and anterior cornual gray matter. At one point in the intermediate gray, there is an open space whose walls are surrounded by layers of small round cells. Sections lower down reveal this as de-

veloped into a well-marked, triangular-shaped cavity lined with cells (see illustration).

Sections in the upper dorsal region show the cavity well developed in the anterior horn of the right side. The right half of the cord is smaller and flattened antero-posteriorly (see cut).

The white matter of the lateral columns is richly studded

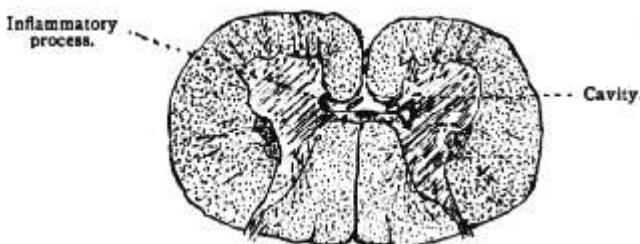


FIG. 2.—Middle cervical region, showing upper limits of lesion. $\times 3$.

with the small colloid-like bodies. There is no marked vascularity. The nerve-cells in the left anterior horn are present only in the antero-internal group. In the region of Clark's column there are a few cells which do not look normal, and there is much vascularity at these places.

The left anterior horn is nearly gone, as shown in the cut. The wall is clean and smooth. At certain levels in

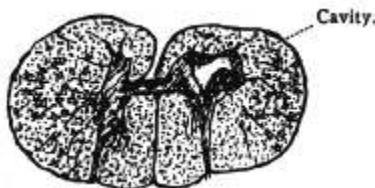


FIG. 3.—Upper dorsal region, showing extent of cavity. $\times 3$.

some sections, however, a cellular infiltration could be seen.

A few anterior cornual cells are left. The posterior horns show a great degree of vascularity, with thick-walled vessels and cellular infiltration around them.

A study of the sections shows that the process began in a congestion, dilatation of vessels, pouring out of cells,

breaking down of surrounding tissue, finally obliteration of vessels and loss of nerve substance. In other words, a slow inflammatory process, beginning in the central gray matter, forming a central focal myelitis, which was gradually extending upwards and downwards. If the patient had lived, other focal points would have doubtless developed, or the process would have extended downward continuously.

The case is one, then, of central focal subacute myelitis, with the formation of cavity.

It is not, strictly speaking, peri-ependymal, for that process is not centred about the central canal.

The case is interesting in connection with the pathology of subacute spinal paralysis.

It also bears upon the question of cavity-building in the cord and of syringomyelia. If anything, it tends to support the views of Hallopeau, Schüppel, Schuele, Eickholt, and others who contend that syringomyelia may be the result of a myelitis.

This above subject has been so thoroughly gone over recently by Oppenheim (*Charité Annalen*, 1886, p. 408) that it need not be taken up here.